Effect of contrasting physical exercise interventions on rapid force capacity of chronically painful muscles

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Andersen LL, Andersen JL, Suetta C, Kjær M, Søgaard K, Sjøgaard G. Effect of contrasting physical exercise interventions on rapid force capacity of chronically painful muscles. *J Appl Physiol* 107: 1413–1419, 2009. First published September 17, 2009; doi:10.1152/japplphysiol.00555.2009.—Rapid force capacity of chronically painful muscles is inhibited markedly more than maximal force capacity and is therefore relevant to assess in rehabilitation settings. Our objective was to investigate the effect of two contrasting types of physical exercise on rapid force capacity, as well as neural and muscular adaptations in women with chronic neck muscle pain. A group of employed women ($n = 42$) with a clinical diagnosis of trapezius myalgia participated in a 10-wk randomized controlled trial; specific strength training of the neck/shoulder muscles, general fitness training performed as leg-bicycling; or a reference intervention without physical activity. Maximal voluntary shoulder abductions were performed at static angles of 35° and 115° with simultaneous recording of electromyography (EMG) in the trapezius and deltoid. Maximal muscle strength and activation (peak torque and peak EMG) as well as rapid muscle strength and activation [rate of torque development (RTD) and rate of EMG rise] were subsequently determined. Trapezius muscle fiber characteristics were determined with ATPase histochemistry. Significant changes were observed only in the specific strength training group. Whereas peak torque increased 18–29% ($P < 0.01$), RTD increased 61–115% ($P < 0.001$). Peak EMG and rate of EMG rise increased correspondingly ($P < 0.05$–0.001), and trapezius type II muscle fibers hypertrophied 20% ($P < 0.001$). In conclusion, rapid force capacity of chronically painful muscles is highly responsive to rehabilitation with specific strength training. The underlying mechanisms were related to both pain reduction and general neuromuscular adaptations to strength training. Potentially, the present method can be a useful clinical screening tool of muscle function in rehabilitation settings.

rate of force development; rehabilitation; myalgia; electromyography; histochemistry; rate of torque development

PAIN AND DISCOMFORT of the musculoskeletal system are a major problem worldwide (35). Neck pain is especially prevalent among female computer users (27). In a recent Danish survey, more than half of female office workers reported frequent neck complaints (16). This is often associated with tenderness and tightness of the upper trapezius muscle, which is clinically characterized as trapezius myalgia (28). In a recent study, two-thirds of female office workers reporting neck pain symptoms were subsequently reported to have a confirmed clinical diagnosis of trapezius myalgia (11). Thus the physiological basis of myalgia as well as adaptations in response to rehabilitation has high clinical relevance.

The debilitating effect of musculoskeletal pain on motor performance is well known (14). Decreased neural drive during maximal voluntary muscle contraction, impaired agonist-antagonist muscle activation patterns, and disturbed control of muscle force steadiness have been reported (14, 32). Particularly, the ability to swiftly activate the muscles and thereby rapidly generate force is impaired in painful muscles (9). We have recently reported markedly reduced rapid force capacity and muscle activation during the initial phase of a maximal voluntary contraction, measured as the rate of torque development and rate of EMG rise, respectively, in women with trapezius myalgia compared with healthy controls, where maximal force capacity was lowered 18%, and rapid force capacity was lowered 54% (9). In painful conditions, the rate of torque development is a more sensitive measure than maximal muscle strength and may consequently be highly responsive to rehabilitation. Although rapid as well as maximal force capacity is influenced by both neural and muscular factors, strength deficits in women with myalgia is not caused by muscular atrophy (12, 13, 31).

Studies have shown strength training to increase rapid force capacity in healthy young (2) as well as frail elderly individuals (38). Increased neural drive and selective type II muscle fiber hypertrophy has been suggested as causal adaptation mechanisms to this type of training (1, 21). We have documented marked pain relief in response to specific strength training in women with chronically painful neck muscles (11). Also, general fitness training which does not directly involve the painful region of the body has been recommended in treating musculoskeletal pain (25). It has been suggested that muscle activity in one part of the body potentially can affect distant muscles as well (33, 45). In support of this, improved endothelial vasodilatory capacity in conduit arteries of nonworking limbs has been observed in response to exercise with the contralateral limb (39), and vascular adaptations in the forearm muscle beds have been found with training of the lower extremities (37). However, no studies have investigated the effect of rehabilitation of pain on rapid force capacity of chronically painful muscles. Whereas general physiological adaptations of strength training are known to increase both rapid and maximal force capacity (2), it can be speculated that reduction of pain per se in response to rehabilitation will increase rapid force capacity. This could be an important component of clinical screening in rehabilitation settings, to more fully assess normalization of muscle function.

The aim of the present study was to investigate the effect of general fitness training and specific strength training on rapid force capacity in women with trapezius myalgia. Furthermore, neural drive and muscle fiber characteristics were assessed to identify their role in the training adaptation. We hypothesized that both types of physical exercise would improve rapid force capacity through decreased pain, but that specific strength training would be superior to general fitness training due to additional effects of general neural and muscular adaptations of strength training.

METHODS

Study Design and Participants

We performed a randomized controlled trial in Copenhagen, Denmark. The study design and procedure of recruitment has been described in detail previously (11). Forty-two women with clinically diagnosed trapezius myalgia participated (44 ± 8 yr, 165 ± 6 cm, 72 ± 15 kg). Exclusion criteria were previous trauma, life-threatening diseases, whiplash injury, cardiovascular diseases, or arthritis in the neck and shoulder. The participants were actively employed and recruited from workplaces with repetitive work tasks, mostly office and computer work. All participants went through a clinical investigation of the neck and shoulder before and after the intervention period. The main criteria for a clinical diagnosis of trapezius myalgia were *1*) chronic or frequent pain in the neck area, *2*) tightness of the upper trapezius muscle, and *3*) palpable tenderness of the upper trapezius muscle (11, 13). We have recently reported isokinetic muscle strength, rate of torque development, and muscle fiber-type composition in the same group of women with trapezius myalgia compared with age-, height-, weight-, and job-matched controls in a cross-sectional study design (9, 12, 13), as well as longitudinal changes in pain and isokinetic muscle strength in response to training intervention (7, 11).

All subjects were informed about the purpose and content of the project and gave written informed consent to participate in the study which conformed to The Declaration of Helsinki and was approved by the Local Ethical Committee (KF 01-138/04). The study qualified for registration in the International Standard Randomised Controlled Trial Number Register: ISRCTN87055459.

Interventions

The participants were randomized into three different intervention groups in a balanced design as described recently (11). The first group, specific strength training, (SST, $n = 18$) performed high-intensity strength training with five dumbbell exercises specifically for the shoulder and neck muscles (1-arm row, shoulder abduction, shoulder elevation, reverse flyes, and upright row) for 20 min three times per week. The specificity and high level of muscle activation of these exercises have been documented previously (10). During each session three of the five different exercises were performed for three sets of each exercise at 8–12 repetitions maximum in a progressive manner. The second group, general fitness training, (GFT, $n = 16$) performed leg-bicycling on an ergometer with relative loadings of 50–70% of the maximal oxygen uptake for 20 min three times per week. The subjects bicycled in an upright position without holding on to the handlebars and with the arms hanging vertically. It was emphasized that the subjects in GFT should relax their shoulders during training. The third group, reference group (REF), received health counseling on group level and individual level with regard to workplace ergonomics, diet, health, relaxation, and stress management for a total of 1 h per week but were not offered any physical training. As a result of the randomization procedure 14 participants were allocated to this group. Unfortunately, withdrawal of six participants who initially stated they would volunteer for the study resulted

in a somewhat smaller REF group ($n = 8$). The subjects were told that all three interventions were considered of equally high standards according to current knowledge.

Dynamometry and Electromyography

A Biodex Medical isokinetic dynamometer (System 3 Pro, Brookhaven R&D Plaza) was used for testing of shoulder abduction at pre- and postintervention. Before the test, EMG electrodes were positioned at the upper trapezius muscle and the mid part of the deltoideus muscle with a bipolar surface EMG configuration (Neuroline 720 01-K, Medicotest A/S, Ølstykke, Denmark) according to standardized procedures (24). Shoulder abductions were performed at two separate static shoulder joint angles (35° and 115°). After warm-up and preconditioning three maximal voluntary contractions (MVCs) were performed at each joint position. Subjects were instructed to contract the muscles as fast and hard as possible (15). All torque and EMG signals were sampled synchronously at 1,000 Hz using a 16-bit analog-to-digital (A/D) converter (DAQ Card-Al-16XE-50, National Instruments) and stored on a laptop for further analysis.

During subsequent off-line analyses, the torque signal was digitally low-pass filtered at 10 Hz and subsequently corrected for the effect of gravity on the subjects arm by adding the passive torque of the arm to the sampled torque signal. EMG parameters were extracted from linear EMG envelopes, which consisted of *1*) high-pass filtering at 10 Hz, *2*) full-wave rectification, and *3*) low-pass filtering at 10 Hz. The filtering algorithms were based on a fourth-order zero phase lag Butterworth filter (43). From the filtered signal the following parameters were extracted.

Maximal muscle activation and torque. For each trial peak torque (PT; N·m) and peak EMG amplitude (PEMG; μV) were determined as the maximal value of the torque-time and EMG-time curve, respectively (Fig. 1). For the statistical analysis the trial with highest PT was used.

Rapid muscle activation and torque. For each trial the rate of torque development (RTD; N·m/s) was determined as the steepest slope over 100 ms of the rising part of the torque-time curve (Fig. 1), i.e., determined as the peak value of a moving window of 100 ms (Δtorque/Δtime). In the same trials the rate of EMG rise (RER) was determined as the steepest slope over 100 ms of the rising part of the EMG-time curve normalized to the individual PEMG (RER;
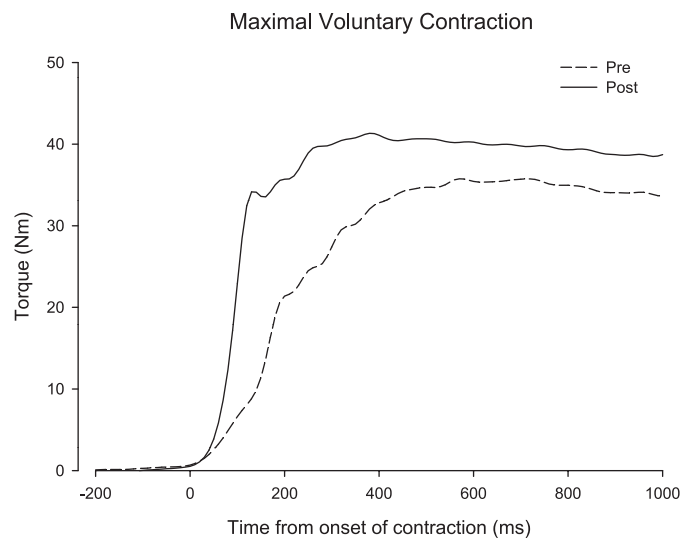

Fig. 1. Torque-time curves of maximal voluntary contractions (35°) from a representative subject before (pre) and after (post) the 10-wk strength training intervention. Notice the markedly steeper slope of the rising part of the torque-time curve (i.e., the rate of torque development) after the intervention.

%PEMG/s). For the statistical analysis, the trial with highest RTD was used.

Muscle Fiber Characteristics

Muscle biopsy samples were obtained at pre- and postintervention from the upper trapezius muscle at the midpoint between the 7th cervical vertebrae and the acromion (13). The posttraining biopsy was obtained within 48–72 h after the last training session. The samples were mounted with Tissue-Tek within 2–3 min, frozen in isopentane precooled with liquid nitrogen, and stored in a freezer at $-80°C$ until further analyses. All biopsy samples were given a unique identification number and blinded. Transverse serial sections (10 μm) of the embedded muscle biopsy were cut in a cryostat (Microm) (22°C) and mounted on glass slides. Standard ATPase analysis was performed after preincubation at pH values of 4.37, 4.61, and 10.30 (17). The biopsy sections were visualized on a computer screen using a Carl Zeiss light microscope (Zeiss Axiolab), a JVC high-resolution color digital camera (JVC, TK-C1381EG), and an eight-bit Matrox Meteor Framegrabber (Matrox Electronic Systems, Quebec, Canada). Quantitative analysis of all muscle samples for fiber cross-sectional area and fiber area percentage was performed using a digital image analysis program (TEMA 1.04, Scanbeam, Hadsund, Denmark) (5). All values are reported for type I and II fibers separately.

Subjective Pain

Pain was rated three times per week during the 10-wk intervention period as "worst pain" experienced since the last session (100 mm VAS scale). These results have been reported previously (11) and were included in the present study for correlation analyses of changes in rapid force capacity.

Statistics

Before the main analyses Shapiro-Wilk testing for normality was performed, showing a normal distribution of the data. Multivariate ANOVA with repeated measures was performed in SAS version 9 (SAS Institute, Cary, NC) using the MIXED procedure to locate differences in the main parameters between the three intervention groups SST, GFT, and REF. Factors included in the model for PT and RTD were group (SST, GFT, and REF), test round (pre- and postintervention), and angle (35 and 115°). For PEMG and RER the factor muscle (deltoid and trapezius) was added to the model. Factors included in the model for muscle fiber cross-sectional area for type I and II fibers were group and test round. For the above analyses, appropriate interactions were tested as well, e.g., group by test round by angle, and group by muscle by test round. When a significant main effect was found, Bonferroni corrected post hoc tests were performed to locate differences. Correlation analyses (Pearson's r) and multiple regression analyses were performed to determine the association between pre- to postintervention changes of the main variables.

Requesting a statistical power of 80% calculations showed that 14 participants should be included in each group for allowing a 15% change with intervention to become significant at the 5% level for the variables and their SDs used in this study with paired analysis. All results are reported as means ± SD.

RESULTS

In response to the 10-wk intervention, significant changes of the present variables were observed only in the specific strength training group (SST).

Maximal Capacity

There was a significant group by test-round effect for PT ($F = 10.7$, $P < 0.0001$). Post hoc tests showed increased PT in SST at both joint angles (Fig. 2A): at 35° from 35 ± 5 to 41 ±
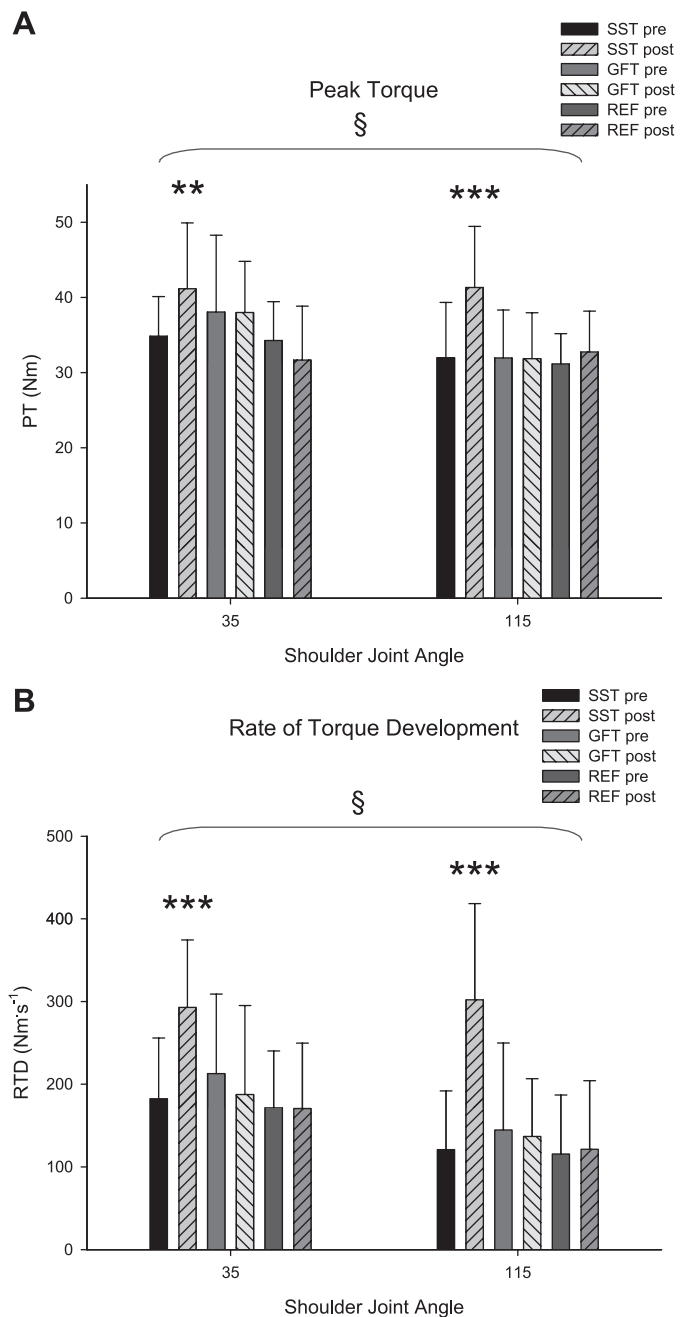

Fig. 2. Peak torque (PT; A) and rate of torque development (RTD; B) at pre- and postintervention for the specific strength training group (SST) as well as the general fitness training and reference group (GFT + REF). A priori hypotheses testing of main effects showed a group by test-round effect for peak torque as well as rate of torque development: §$P < 0.0001$. Post hoc analyses showed a significant effect only for SST: Post > pre, **$P < 0.01$, ***$P < 0.001$.

9 N·m ($P < 0.01$) and at 115° from 32 ± 7 to 41 ± 8 N·m ($P < 0.001$).

There was a significant group by test-round by muscle effect for PEMG ($F = 5.60$, $P < 0.001$). Post hoc tests showed increased trapezius PEMG in SST at both joint angles (Fig. 2A), at 35° from 682 ± 429 to 976 ± 499 μV ($P < 0.05$) and at 115° from 824 ± 380 to 1,109 ± 405 μV ($P < 0.05$), whereas no significant change occurred in deltoideus PEMG (Table 1).

Table 1. *Peak EMG amplitude and rate of EMG rise at pre- and postintervention for the specific strength training group as well as the general fitness training and reference groups*

	PEMG, μV^a		RER, %PEMG/s^b	
	Trapezius	Deltoideus	Trapezius	Deltoideus
	35°			
REF				
Pre	777±370	329±147	512±317	531±237
Post	796±254	384±136	569±266	464±123
GFT				
Pre	577±355	391±208	578±243	465±219
Post	617±313	470±288	417±231	435±265
SST				
Pre	682±429	415±292	534±215	470±148
Post	976±499^c	529±298	641±174^c	654±187^d
	115°			
REF				
Pre	963±486	421±225	401±263	435±219
Post	966±201	540±201	408±216	387±145
GFT				
Pre	752±415	507±278	368±182	381±195
Post	774±506	590±356	356±131	404±166
SST				
Pre	824±380	523±271	373±162	368±191
Post	1109±405^c	579±229	590±157^e	637±185^e

Values are means ± SD. PEMG, peak EMG amplitude; RER, rate of EMG rise; REF, reference group; GFT, general fitness training group; SST, specific strength training group. 35° and 115° refer to joint angles. A priori hypotheses testing of main effects showed a group by muscle by test-round effect ($^a P < 0.001$) for PEMG, and a group by test-round effect ($^b P < 0.001$) for RER. Post hoc analyses showed a significant effect only for SST: post > pre, $^c P < 0.05$, $^d P < 0.01$, $^e P < 0.001$, respectively.

The interaction effect of joint angle was not significant.

Rapid Capacity

There was a significant group by test-round effect for RTD ($F = 22.0$, $P < 0.0001$). Post hoc tests showed increased RTD in SST at both joint angles (Fig. 2*B*): at 35° from 182 ± 73 to 293 ± 82 N·m/s ($P < 0.001$) and at 115° from 121 ± 71 to 302 ± 116 N·m/s ($P < 0.001$).

There was a significant group by test-round effect for RER ($F = 16.0$, $P < 0.0001$). Post hoc tests showed increased RER in SST at both joint angles and in both muscles (Table 1): at 35° from 534 ± 215 to 641 ± 174 μV ($P < 0.05$) for the trapezius and from 470 ± 148 to 654 ± 187 μV ($P < 0.01$) for the deltoid, and at 115° from 373 ± 162 to 590 ± 157 μV ($P < 0.001$) for the trapezius and from 368 ± 191 to 637 ± 185 μV ($P < 0.001$) for the deltoideus (Table 1).

The interaction effects of angle and muscle were nonsignificant, i.e., changes in response to the intervention were not significantly different across joint angles (RTD and RER) and muscles (RER).

Muscle Fiber Characteristics

There was a significant group by test-round effect for type II muscle fiber size ($F = 4.7$, $P < 0.05$). Post hoc tests showed hypertrophy of type II muscle fibers in SST from 3,439 ± 1,331 to 4,133 ± 1,145 μm^2 (~20%, $P < 0.001$). There was a tendency for a group by test-round effect for type I muscle

Table 2. *Muscle fiber cross-sectional area and fiber type area percentage at pre- and postintervention for the specific strength training group as well as the general fitness training and reference groups*

	Area, μm^2		Area Percentage, %	
	Type I	Type II†	Type I	Type II
REF				
Pre	4,941±399	3,334±489	75±9	25±9
Post	5,299±1,134	3,471±684	75±11	26±11
GFT				
Pre	5,555±1,201	3,794±796	77±9	24±9
Post	5,108±1,030	3,612±1,070	79±9	22±9
SST				
Pre	5,043±1,294	3,439±1,331	76±11	25±9
Post	5,516±1,188	4,133±1,145*	75±12	26±11

Values are means ± SD. A priori hypotheses testing of main effects showed a group by test-round effect (†$P < 0.01$) for type II muscle fiber cross-sectional area. Post hoc analyses showed a significant effect only for SST: Post > pre, *$P < 0.001$.

fiber size ($F = 3.0$, $P = 0.06$). Likewise, post hoc tests showed a tendency for hypertrophy of type I fibers in SST from 5,043 ± 1,294 to 5,516 ± 1,188 μm^2 (~9%, $P = 0.09$). The area percentage of type I and II fibers remained statistically unchanged (Table 2).

Association Between Variables

The pre- to postintervention change in rate of torque development was significantly correlated to the change in rate of EMG rise, peak torque, and pain, but not significantly to the change in type II fiber cross-sectional area (Table 3). When all these variables were combined in a multiple regression analyses, the explained variance for the pre- to postintervention change in rate of torque development was 75% at 35° of shoulder abduction ($P < 0.0001$) and 83% at 115° of shoulder abduction ($P < 0.0001$).

DISCUSSION

These results demonstrated increased rapid force capacity of chronically painful muscles in response to rehabilitation with specific strength training. Both neural and muscle cellular adaptation mechanisms were evident. In contrast, neither general fitness training nor a reference intervention without spe-

Table 3. *Correlation coefficients (Pearson's r) between pre- and postintervention changes in rate of torque development, pain intensity, peak torque, rate of EMG rise, and type II fiber cross-sectional area*

	RTD	
	35°	115°
Pain	−0.17	−0.42*
PT	0.53*	0.76*
RER deltoid	0.68*	0.81*
RER trapezius	0.69*	0.63*
Type II fiber CSA	0.03	0.17

Values are means ± SD. RTD, rate of torque development; PT, peak torque; RER, rate of EMG rise; CSA, cross-sectional area. *$P < 0.001$–0.05. Pain data from a previously published article on the same study group was used solely for this correlation analysis (11).

cific physical training significantly altered any of these variables.

In the present group of subjects, we have previously shown in a cross-sectional study a markedly lowered rapid force capacity and neural drive during the initial phase of a maximal voluntary contraction (9). Further, we have also reported decreased subjective pain intensity of 79% in response to 10 wk specific strength training, but not in response to leg-bicycling or reference intervention (11). The present study elaborates on these findings by showing that specific strength training counteracts inhibition of rapid muscle activation leading to significant increase of rapid force capacity. Actually, the rate of torque development and rate of EMG rise were raised to levels previously reported in healthy matched controls (9).

The present results suggest that the underlying adaptation mechanisms of increased rapid force capacity are multifactorial. In nonpainful conditions, rapid force capacity is influenced particularly by central neural drive (2, 20, 40), muscle fiber characteristics (23), and maximal muscle strength (6). Although such adaptations occurred in the present study, the markedly increased rate of EMG rise suggests that neural adaptations were the predominant mechanism. Thus enhancement of rapid force capacity occurred in parallel with increased rate of EMG rise. Whereas this has been reported in healthy subjects (2), the present study documents a significant correlation between changes in these variables in response to the intervention. Increased motoneuron firing frequency and recruitment of high-threshold motor units during the very initial phase of muscle contraction may explain increased rate of EMG rise (30, 40).

Rapid force capacity of healthy muscles is moderately to strongly related to maximal muscle strength (6, 34). The present study extends these findings, by showing a moderate to strong correlation between changes in maximal muscle strength and rapid force capacity with rehabilitation of painful muscles.

Selective hypertrophy of type II muscle fibers was observed in the present study. Cross-bridge cycling rate of type II fibers markedly exceed that of type I fibers, which is especially important for rapid development of muscle force (22). While selective hypertrophy of type II muscle fibers theoretically could contribute to increased rapid force capacity, this was not confirmed by the present correlation analyses. Although the inherent variance associated with muscle biopsies makes correlation analyses difficult in small sample sizes, the present finding suggests that type II muscle fiber hypertrophy was not the predominant mechanism of increased rapid force capacity. Altogether, several of the above-mentioned adaptation mechanisms may have acted in concert to increase rapid force capacity.

While part of the observed adaptations could be caused by general effects of strength training, others may be mediated by pain reduction. Selective hypertrophy of type II muscle fibers has previously been reported in response to strength training of healthy as well as myalgic muscles (5, 29). Likewise, increased maximal voluntary static strength of 20–30%, as observed in the present study, is expected in response to high-intensity strength training intervention (3, 8). Altogether, the magnitude of these adaptations in the present study is generally within the expected range of a strength training intervention in healthy individuals. However, the 61–115% increase of rapid force capacity markedly exceeds that previously reported in young (2) as well as frail elderly subjects (38). A pain-related low initial rapid force capacity in women with myalgia compared with healthy matched individuals (9) may explain the high responsiveness to strength training. Thus intensity of pain was more than halved in response to specific strength training (11), which may have boosted rapid force capacity beyond adaptations typically found in response to strength training of healthy muscles.

Although not significantly different, rapid force capacity increased numerically more at the most abducted shoulder joint angle, i.e., increase of 115% at 115° vs. 61% at 35°. Interestingly, the most pronounced deficit of rapid force capacity was found at this angle in the baseline study of the same individuals (9). Further, in the present study a significant correlation between changes in pain and rapid force capacity was found only at 115°. Together these findings support that reduction of pain in response to the intervention augmented rapid muscle activation, especially at the most abducted joint position with the highest level of pain inhibition. As a complementary explanation, it has been suggested that pain-related beliefs, such as fear avoidance due to longstanding pain, are more important determinants of disability in patients with musculoskeletal disorders than intensity of pain per se (18). Thus the belief that rapid movement exacerbates pain (4, 19, 36) could potentially limit neural drive. Therefore, changes in pain-related beliefs during the course of training, and not only reduction of pain per se, may have contributed to the present findings. Although fear avoidance was not quantified in the present study, reduction of fear avoidance as training loads progressively get heavier and pain decreases seems plausible.

Although leg bicycling has previously been reported to result in acute pain reduction (11), no significant influence on rapid force capacity was seen in the present study, likely because bicycling was not performed immediately before testing. Future studies should evaluate the impact of acute pain-relief, e.g., by bicycling or local injection of analgesics, on rapid force capacity of chronically painful muscles.

The findings of this study demonstrated that rapid force capacity was normalized in response to 10 wk of specific strength training. Rapid force capacity appears to be a more sensitive measure than maximal muscle strength in response to rehabilitation, suggesting this would be a useful clinical monitoring tool.

A limitation of this study is the lack of a healthy group of females undergoing the same type of intervention as a control group. Future studies are needed to compare the effect of strength training on rapid force capacity in subjects with and without initial pain on gains in rapid vs. maximal force capacity. Another limitation is the modest sample size due to initial dropout of participants in REF. However, since no change occurred either in REF or GFT, both of these groups may be considered as reference interventions to SST. It should be noted that subjective pain scores were used in the present study, and that this may not always correlate to objective pathology. However, subjective pain scores (26) provide the most accepted method to track changes in neck/shoulder pain in response to training interventions (41, 42, 44).

Conclusion

Rapid force capacity of chronically painful muscles does respond to rehabilitation with specific strength training. The underlying mechanisms were related to both pain reduction and general neuromuscular adaptations to strength training. Potentially, this method can be added to the umbrella of clinical screening tools used for assessment of muscle function in rehabilitation settings.

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